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Research paper

A time series analysis to investigate the effect of inhalation of aldehyde C10 on the human EEG activity

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A R T I C L E I N F O

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A B S T R A C T

Introduction: Decanal (C10) is an important aldehyde, extensively used to enhance floral and citrus notes in various perfumery products. It is well-known that the human electroencephalographic (EEG) activity is highly susceptible to change due to the exposure of fragrances. However, the EEG findings exhibit non-stationary behavior in terms of analysis and recording time. Hence, the present study aimed to investigate the effect of inhalation of aldehyde C10 on the human EEG activity with respect to time series analysis.

Methods: Twenty healthy volunteers (10 men and 10 women) participated in the EEG study. The EEG data were recorded from 8 channels according to the International 10–20 System. The EEG readings were analyzed for every second by splitting the total 30 s data during the no odor and C10 odor exposures.

Results: The exposure of C10 odor produced significant changes ($p < 0.05$) in all the absolute waves at a certain time during the time series analysis. The results revealed that all absolute waves significantly decreased during the first 13 s period of time due to the exposure of C10 odor. After that, absolute alpha, absolute slow alpha, and absolute fast alpha markedly increased. Furthermore, the exposure to C10 appears to mainly affect the frontal regions, especially the left frontal region (F3) compared with other regions.

Conclusion: Our data suggest that the EEG activity of C10 odor is highly unstable in the time series analysis, thereby analysis time could play a key role in the EEG response to olfactory stimulation.

1. Introduction

Historically, fragrances have been used for enhancing cognitive performances and other behavioral activities of the human brain [1,2]. Volatile components (terpenes and their oxygenated derivatives) are considered particularly as primary sources of fragrances for perfumery and cosmetic products. Aldehydes are one of the important floral fragrant compounds as in a legendary perfume Chanel No. 5, which is mainly perceived as a pleasant smell by humans. Aldehydes are organic compounds which incorporates a carbonyl functional group (C═O) and widely found in nature. In the environment, aldehydes are derived from natural as well as anthropogenic sources. They can be formed endogenously by lipid peroxidation, autoxidation of carbohydrate or ascorbate, amine oxidases, etc [3].

Among the various aldehydes, octanal (C8), nonanal (C9) and decanal (C10) aldehydes are important bioactive fragrant components used in a variety of fragrance types, including green-floral and aldehydic [4]. In these, decanal (C10) is a widely used fragrant component in perfume industries, especially in blossom fragrances. It has characteristic odors of waxy, aldehydic, orange and citrus. The aldehyde C10 is mainly used for floral as well as citrus like fragrances in perfumery products and occurs widely in various essential oils, including neroli oil, kesum oil, coriander oil, different citrus peel oils, and various Mediterranean woody species. It is also a major component of buckwheat odor [5,6]. It is well-known that fragrances exhibit various positive psychophysiological conditions of the human such as emotions, thoughts, and memory [7].

The human being is able to recognize different types of fragrances via orthonasal and retronasal inhalations. The brain functional changes induced by fragrance inhalation are mainly associated with the modulation of neuronal electrical activity via the olfactory nervous system [8]. Different brain waves are considered to have relationships with particular brain functions. Previously, several studies have reported that the electroencephalograph (EEG) technique is commonly used to clarify the psychophysiological effects of fragrances [9–11]. This technique records the fluctuations of electrical waveforms on the scalp. In

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addition, the published reports have clearly indicated that the human EEG activity is highly susceptible to change during the exposure of odors [12]. The EEG recording is quite simple, non-invasive and could serve as an objective tool for determining the brain state. In addition, it is easy to obtain results in a short time without an active cooperation of subjects [13]. EEG data has been exhibited to show non-stationary behavior in a number of contexts. According to different authors, the recording time for evaluating the effect of odors may vary from a few seconds to minutes [2,14]. Hence, time series analysis of EEG data is a useful way to understand a specific trend in the EEG readings. It is an ordered sequence of values of a variable at uniformly spaced time intervals.

Furthermore, there is no study in relation to the inhalation of C10 aldehyde on human EEG activity. Based on the above, the present study was carried out to investigate the effect of inhalation of aldehyde C10 odor on human EEG activity according to time series analysis.

2. Materials and methods

2.1. Material

Decanal (CAS No. 112-31-2) was purchased from Sigma (St. Louis, MO, USA). The purchased chemical was stored at 4 °C until used for EEG study.

2.2. Subjects

The present study followed the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The study protocol was approved by the ethics committee from the Kangwon National University, Chuncheon, Republic of Korea (IRB No. KWNUIRB-2017-05-001-003). Twenty healthy subjects (10 men and 10 women) aged 20–30 years participated in this study. The mean ages of men and women were 22.7 ± 2.8 and 21.4 ± 3.9, respectively. The inclusion criteria for the subjects were non-smokers and right-handed without any abnormalities in the olfaction. None of the subjects had olfactory diseases or abused drugs. Alcohol consumption or medications was prohibited from 2 days before the experiment. There were no statistically significant differences between men and women. The odor of familiar fragrances such as rose, citrus, and lavender was evaluated by the volunteer subjects. For this purpose, one drop of each essential oil was separately added on a commercial odor-strip and placed at 5 cm from the nose of the subject. The subjects who unable to recognize the familiar fragrance types were excluded from this study.

2.3. Experimental design

A single group pretest and posttest experimental design was used in the study (20 subjects). During the EEG recordings, the subjects were instructed to sit quietly, keep their eyes closed and breathe normally but to remain awake. After the EEG recordings, the subjects were asked to give their preference and impression of the fragrance of C10.

2.4. EEG recordings

The EEG readings were recorded using a QEEG-8 system (LXE3208, LAXTHA Inc., Daejeon, Republic of Korea). The EEG data were recorded from 8 channels located on the scalp at left prefrontal (Fp1), right prefrontal (Fp2), left frontal (F3), right frontal (F4), left temporal (T3), right temporal (T4), left parietal (P3), and right parietal (P4) regions according to the International 10–20 System previously described by Kim et al. [9].

2.5. Fragrance administration

The aldehyde, C10 was used as the fragrance stimulus. The EEG recording room was maintained with a constant temperature (23 °C) and humidity (50%). The undiluted C10 (10 μl) was added on the perfumer's paper strip and then placed about 3 cm in front of the subject's nose. The baseline EEG readings were recorded in an eye-closed state for each condition. Firstly, EEG was recorded 45 s during the no odor exposure, then EEG was recorded 45 s during the exposure of C10 odor. The interval time between these two conditions was 3 min.

Table 1
EEG power spectrum indices used in this study.

S. No.	EEG Indices	The full name of the EEG power spectrum indices	Wavelength range (Hz)
1	AT	Absolute theta	4 ~ 8
2	AA	Absolute alpha	8 ~ 13
3	AB	Absolute beta	13 ~ 30
4	AG	Absolute gamma	30 ~ 50
5	ASA	Absolute slow alpha	8 ~ 11
6	AFA	Absolute fast alpha	11 ~ 13
7	ALB	Absolute low beta	12 ~ 15
8	AMB	Absolute mid beta	15 ~ 20
9	AHB	Absolute high beta	20 ~ 30
10	RT	Relative theta	(4 ~ 8) / (4 ~ 50)
11	RA	Relative alpha	(8 ~ 13) / (4 ~ 50)
12	RB	Relative beta	(13 ~ 30) / (4 ~ 50)
13	RG	Relative gamma	(30 ~ 50) / (4 ~ 50)
14	RSA	Relative slow alpha	(8 ~ 11) / (4 ~ 50)
15	RFA	Relative fast alpha	(11 ~ 13) / (4 ~ 50)
16	RLB	Relative low beta	(12 ~ 15) / (4 ~ 50)
17	RMB	Relative mid beta	(15 ~ 20) / (4 ~ 50)
18	RHB	Relative high beta	(20 ~ 30) / (4 ~ 50)
19	RST	Ratio of SMR to theta	(12 ~ 15) / (4 ~ 8)
20	RMT	Ratio of mid beta to theta	(15 ~ 20) / (4 ~ 8)
21	RSMT	Ratio of SMR ~ mid beta to theta	(12 ~ 20) / (4 ~ 8)
22	RAHB	Ratio of alpha to high beta	(8 ~ 13) / (20 ~ 30)
23	SEF50	Spectral edge frequency 50%	4 ~ 50
24	SEF90	Spectral edge frequency 90%	4 ~ 50
25	ASEF	Spectral edge frequency 50% of alpha	8 ~ 13

2.6. Data analysis

The mean power spectrum values [microvolt square (μV^2)] were calculated for 25 EEG indices (Table 1) including absolute and relative power spectra of theta, alpha, beta, and gamma waves [15]. Fast Fourier transform was used to measure the mean power values (μV2). In the total 45 s of EEG recording, we selected the first 30 s of EEG raw data for the analysis. We analyzed the EEG data for every second by splitting the total 30 s EEG data. Statistical analysis was performed using SPSS statistical package 23 (SPSS, Inc., Chicago, IL, USA). The EEG power spectrum values during the inhalation of no odor and C10 odor were analyzed by a paired Student's *t*-test and the p value < 0.05 was considered significant. The t-Mapping of EEG power spectra changes was constructed by the Telescan software package (LXSMD61, LAXTHA Inc., Daejeon, Republic of Korea).

3. Results

The olfactory stimulation effect of inhalation of undiluted aldehyde C10 on EEG activity was investigated in this study. We analyzed EEG changes during no odor and C10 odor exposures for every second by splitting the total 30 s EEG data. Out of 25 EEG indices, 9 absolute indices such as absolute theta (AT), absolute alpha (AA), absolute beta (AB), absolute gamma (AG), absolute slow alpha (ASA), absolute fast alpha (AFA), absolute low beta (ALB), absolute mid beta (AMB), and absolute high beta (AHB) were included in the analysis. Table 2 shows the significant changes of EEG power spectrum values during no odor and C10 odor exposures according to time series analysis. According to the EEG time series analysis, the exposure of C10 odor produced significantly different EEG power spectrum changes at different brain

Table 2
Significant changes of EEG power spectrum values at different time series during no odor and C10 odor exposures.

Time (s)	EEG Indices	Site	No odor inhalation (μV^2)	C10 odor inhalation (μV^2)	t-test	*p* value*
6 ~ 7	AMB	F3	8.2124 ± 1.139	5.8006 ± 0.805	2.382	0.028
		F4	6.7492 ± 0.850	4.9640 ± 0.572	2.651	0.016
	AG	Fp1	9.9255 ± 1.146	7.7770 ± 1.097	2.102	0.049
7 ~ 8	ASA	P3	57.2825 ± 12.618	38.9928 ± 9.443	2.356	0.029
	AT	P4	25.8167 ± 5.061	15.1191 ± 2.278	2.348	0.030
		T4	15.9907 ± 3.262	9.6612 ± 1.600	2.156	0.044
	AA	P3	64.9300 ± 14.105	46.4312 ± 9.653	2.099	0.049
8 ~ 9	AA	T3	33.5684 ± 7.516	22.4929 ± 3.960	2.589	0.018
	ASA	T3	28.5238 ± 6.952	18.6194 ± 3.398	2.334	0.031
	AFA	T3	12.8180 ± 2.681	8.5705 ± 1.491	2.564	0.019
9 ~ 10	AA	P3	72.0094 ± 18.221	42.1010 ± 10.037	2.318	0.032
	ASA	P3	63.9246 ± 17.628	36.8357 ± 9.744	2.259	0.036
	AT	T4	18.0804 ± 3.962	11.4483 ± 1.962	2.138	0.046
12 ~ 13	AA	F4	67.9684 ± 14.642	44.9909 ± 9.632	2.458	0.024
		Fp2	51.6642 ± 10.665	37.7460 ± 7.481	2.113	0.048
	ASA	F4	61.7747 ± 14.695	41.5105 ± 9.606	2.106	0.049
13 ~ 14	AA	F4	53.6288 ± 9.292	37.0096 ± 5.286	2.644	0.016
		Fp2	44.4451 ± 7.633	31.0424 ± 4.591	2.564	0.019
		P4	49.8133 ± 9.089	32.0621 ± 5.181	2.430	0.025
		P3	56.3988 ± 11.174	34.3043 ± 4.819	2.358	0.029
		F3	55.2476 ± 9.161	37.8680 ± 5.157	2.285	0.038
		Fp1	44.0420 ± 7.615	31.4006 ± 4.396	2.165	0.046
	ASA	P3	47.7617 ± 10.244	26.7331 ± 4.770	2.587	0.018
		P4	39.9391 ± 7.841	23.2314 ± 4.656	2.561	0.019
		F4	46.8484 ± 8.849	32.9406 ± 5.207	2.475	0.023
		F3	48.8388 ± 8.793	33.1817 ± 5.067	2.230	0.038
		Fp2	38.4598 ± 7.300	27.9963 ± 4.508	2.131	0.046
	ALB	Fp2	8.0243 ± 1.509	4.1173 ± 0.570	2.385	0.028
15 ~ 16	ALB	T4	4.6877 ± 0.670	2.5008 ± 0.360	2.953	0.008
16 ~ 17	AHB	F4	6.5191 ± 0.831	5.2974 ± 0.636	2.104	0.049
17 ~ 18	AHB	F3	9.1458 ± 1.228	6.6828 ± 0.593	2.156	0.044
	AMB	F3	7.5403 ± 1.081	5.5869 ± 0.477	2.134	0.046
18 ~ 19	ALB	T4	6.4192 ± 1.466	3.324 ± 0.341	2.198	0.041
	AG	Fp1	6.7972 ± 0.743	8.9215 ± 1.240	−2.135	0.046
19 ~ 20	AMB	P3	5.4716 ± 0.649	8.2285 ± 1.579	−2.214	0.039
21 ~ 22	AHB	Fp1	5.9192 ± 0.581	8.7631 ± 1.087	−3.112	0.006
		Fp2	5.7070 ± 0.816	8.2532 ± 1.168	−2.895	0.009
		F3	5.7102 ± 0.481	8.1700 ± 1.225	−2.372	0.028
		F4	5.0788 ± 0.721	6.5907 ± 0.921	−2.378	0.028
	ALB	F3	5.0157 ± 0.621	9.5567 ± 1.280	−3.093	0.006
		F4	4.9597 ± 0.645	8.7863 ± 1.434	−2.713	0.014
		Fp2	4.4686 ± 0.516	7.6374 ± 1.239	−2.374	0.028
	AFA	F3	9.2697 ± 2.09	20.2843 ± 3.916	−3.078	0.006
		F4	10.0755 ± 1.972	17.0146 ± 3.495	−2.124	0.047
21 ~ 22	AB	F3	12.3997 ± 1.129	17.3331 ± 1.662	−2.923	0.009
		F4	11.2669 ± 1.454	15.5090 ± 1.814	−2.159	0.044
		Fp2	11.7369 ± 1.558	15.5051 ± 1.671	−2.263	0.044
	AA	T3	20.0190 ± 4.220	30.7232 ± 6.118	−2.374	0.028
		F3	33.2826 ± 7.669	51.1288 ± 11.180	−2.224	0038
	ASA	T3	16.7221 ± 4.071	26.4641 ± 5.723	−2.257	0.036
22 ~ 23	ALB	F3	4.9896 ± 0.766	7.1687 ± 1.057	−2.429	0.025
23 ~ 24	AMB	F3	4.6294 ± 0.721	8.1899 ± 1.445	−2.414	0.026
		F4	3.9469 ± 0.630	6.8486 ± 0.970	−2.679	0.015
		Fp1	4.3686 ± 0.608	7.1595 ± 1.067	−2.285	0.034
		Fp2	3.5416 ± 0.510	6.5939 ± 0.991	−2.610	0.017
	AHB	P4	6.0309 ± 0.794	7.4475 ± 0.849	−2.198	0.041
	AA	T4	13.2868 ± 2.225	18.8106 ± 3.794	−2.179	0.042
24 ~ 25	AMB	P3	9.4065 ± 1.741	5.4286 ± 0.738	2.431	0.025
		F3	8.4930 ± 1.594	4.8927 ± 0.506	2.399	0.027
25 ~ 26	ALB	Fp1	5.8226 ± 1.092	9.91835 ± 1.777	−2.667	0.015
27 ~ 28	ALB	Fp1	7.4592 ± 0.902	5.4563 ± 0.709	3.227	0.004
		Fp2	6.6535 ± 0.875	5.1176 ± 0.680	2.434	0.025
	AMB	F3	9.6618 ± 1.444	6.0245 ± 0.846	2.643	0.016
		F4	8.1441 ± 1.241	5.2794 ± 0.790	2.124	0.047
		Fp1	8.2736 ± 1.087	5.1109 ± 0.673	2.441	0.025
		P3	8.7040 ± 1.509	5.8510 ± 0.824	2.243	0.037
	AB	F3	21.0605 ± 3.095	14.9596 ± 1.484	2.662	0.017
		Fp1	19.6644 ± 2.484	13.4600 ± 1.177	2.585	0.018
		F4	17.7773 ± 2.714	12.4336 ± 1.373	2.312	0.032
		P3	22.0850 ± 3.089	17.0941 ± 2.073	2.368	0.029
29 ~ 30	AT	Fp1	22.7334 ± 2.956	16.3276 ± 2.548	2.127	0.047
		Fp2	22.1628 ± 2.846	14.8205 ± 1.913	2.945	0.008
		F4	28.8708 ± 4.836	18.7353 ± 2.197	2.291	0.034

(*continued on next page*)

Table 2 (*continued*)

Time (s)	EEG Indices	Site	No odor inhalation (μV^2)	C10 odor inhalation (μV^2)	t-test	*p* value*
31 ~ 32	AFA	T3	6.7998 ± 1.315	11.0558 ± 1.604	−2.539	0.020
	AT	P3	23.8442 ± 4.016	18.4420 ± 2.950	2.324	0.031
32 ~ 33	ASA	Fp1	25.6643 ± 5.123	35.4493 ± 7.134	−2.704	0.014
		F3	32.9264 ± 6.674	45.9028 ± 8.779	−2.525	0.021
		Fp2	26.5527 ± 5.665	34.9311 ± 6.993	−2.236	0.038
	AA	F3	36.6221 ± 6.639	48.3807 ± 8.930	−2.498	0.022
		Fp1	29.2620 ± 5.351	37.8193 ± 7.283	−2.347	0.030
		Fp2	29.1933 ± 5.825	37.1964 ± 7.044	−2.130	0.046
	ALB	Fp1	6.5935 ± 1.119	4.0769 ± 0.454	2.233	0.038
33 ~ 34	AFA	F4	8.9180 ± 1.603	16.4875 ± 3.703	−2.948	0.008
		F3	10.0714 ± 1.845	16.8827 ± 3.737	−2.556	0.019
		Fp1	8.9851 ± 1.467	15.1711 ± 3.507	−2.299	0.033
		Fp2	7.8837 ± 1.341	14.8734 ± 3.211	−2.917	0.009

AT, absolute theta; AA, absolute alpha; AB, absolute beta; AG, absolute gamma; ASA, absolute slow alpha; AFA, absolute fast alpha; ALB, absolute low beta; AMB, absolute mid beta; AHB, absolute high beta; Fp1, left prefrontal; Fp2, right prefrontal; F3, left frontal; F4, right frontal; T3, left temporal; T4, right temporal; P3, left parietal; P4, right parietal. Number of subjects – 20. *Significant difference ($p < 0.05$).

Table 3
Significant increase and decrease of absolute brain waves at different time series during the exposure of C10 odor.

Time (s)	AT	AA	AB	AG	AFA	ASA	ALB	AMB	AHB
5~6									
6~7				↓				↓	
7~8	↓	↓				↓			
8~9		↓			↓	↓			
9~10	↓	↓				↓			
10~11									
11~12									
12~13		↓				↓			
13~14		↓				↓	↓		
14~15									
15~16							↓		
16~17									↓
17~18								↓	↓
18~19				↑			↓		
19~20								↑	
20~21									
21~22		↑	↑		↑	↑	↑		↑
22~23							↑		
23~24		↑						↑	↑
24~25								↓	
25~26							↑		
26~27									
27~28			↓				↓	↓	
28~29									
29~30	↓								
30~31									
31~32	↓				↑				
32~33		↑				↑	↓		
33~34					↑				
34~35									

AT, absolute theta; AA, absolute alpha; AB, absolute beta; AG, absolute gamma; ASA, absolute slow alpha; AFA, absolute fast alpha; ALB, absolute low beta; AMB, absolute mid beta; AHB, absolute high beta. Arrows show the decrease and increase of absolute brain waves during the inhalation of C10 odor.

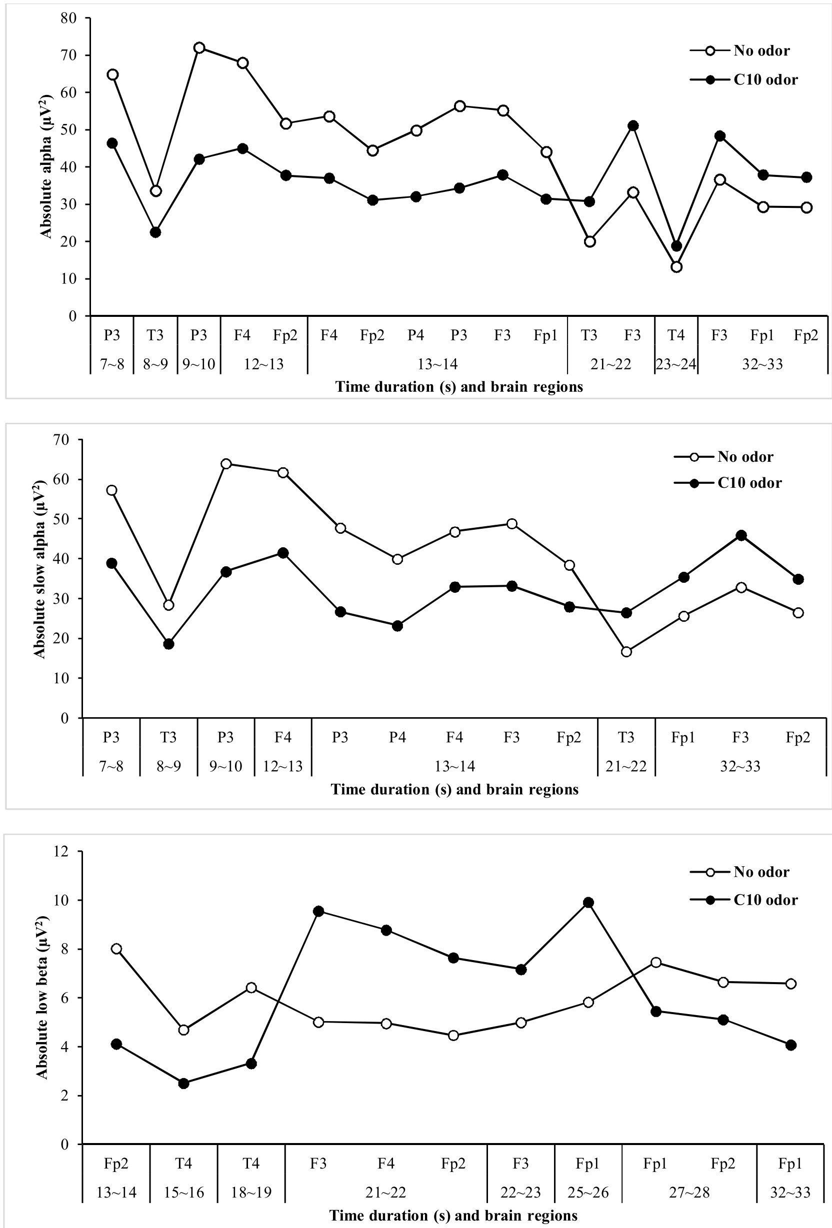

Fig. 1. Significant changes of absolute alpha, absolute fast alpha, and absolute low beta power spectra during no odor and C10 odor exposures according to time series analysis. Fp1, left prefrontal; Fp2, right prefrontal; F3, left frontal; F4, right frontal; T3, left temporal; T4, right temporal; P3, left parietal; P4, right parietal.

regions. However, there is no significant change in the absolute waves at 5 ~ 6, 10 ~ 12, 14 ~ 15, 20 ~ 21, 26 ~ 27, 28 ~ 29, 30 ~ 31 and 34 ~ 35 s during the exposure of C10 odor. From 6–18 s, all absolute brain waves (except absolute beta) significantly decreased at a certain time during the exposure of C10 odor when compared with no odor exposure. Out of 30 time series analysis data (from 5 ~ 35 s), significant differences of AA and ALB waves were observed 8 times followed by AFA (7 times) than other absolute waves (Table 3; Fig. 1). The t-mapping of brain wave changes evidently expressed the alteration of EEG activity due to the exposure of C10 (Figs. 2–4).

Among different absolute waves, AT wave activity significantly decreased at 7 ~ 8 s (25.8167 – 15.1191 μV^2 at P4 and 15.9907 – 9.6612 μV^2 at T4), 9 ~ 10 s (18.0804 – 11.4483 μV^2 at T4), 29 ~ 30 s (22.7334 - 16.3276 μV^2 at Fp1, 22.1628 - 14.8205 μV^2 at Fp2 and 28.8708 -18.7353 μV^2 at F4) and 31 ~ 32 s (23.8442 - 18.4420 μV^2 at P3). AB significantly increased at 21 ~ 22 s (Fp2, F3 and F4) and decreased at 27 ~ 28 s (Fp1, F3, F4, and P4). In addition, AG wave activity significantly decreased at 6 ~ 7 s (9.9255 – 7.7770 μV^2 at Fp1) and increased at 18 ~ 19 s (6.7972–8.9215 μV^2 at Fp1). ALB wave significantly decreased from 13 ~ 14 s to 18 ~ 19 s and from 27 ~ 28 s to 32 ~ 33 s. In between, ALB significantly increased from 21 ~ 22 s to 25 ~ 26 s. AHB also significantly decreased at 16 ~ 18 s and increased from 21 ~ 22 s to 23 ~ 24 s. AA and ASA waves significantly decreased at similar time series from 7 ~ 8 s to 13 ~ 14 s. In particular, AA (Fp1, Fp2, F3, F4, P3, and P4) markedly decreased in the most number of brain regions (except temporal regions) during the period of 13 ~ 14 s. Further, significant increases of AA, ASA, and AFA were observed from 21 ~ 22 s to 33 ~ 34 s. However, there was no significant change in the absolute alpha waves during the period of 14 ~ 21 s (Table 2).

EEG analysis time period during 21 ~ 22 s exhibited significant changes in more number of indices such as AA, AB, AFA, ASA, ALB, and AHB due to the exposure of C10 odor when compared with other analysis times. The results revealed that the odor of C10 mainly affected absolute wave activities at frontal regions (F3 and F4) followed by

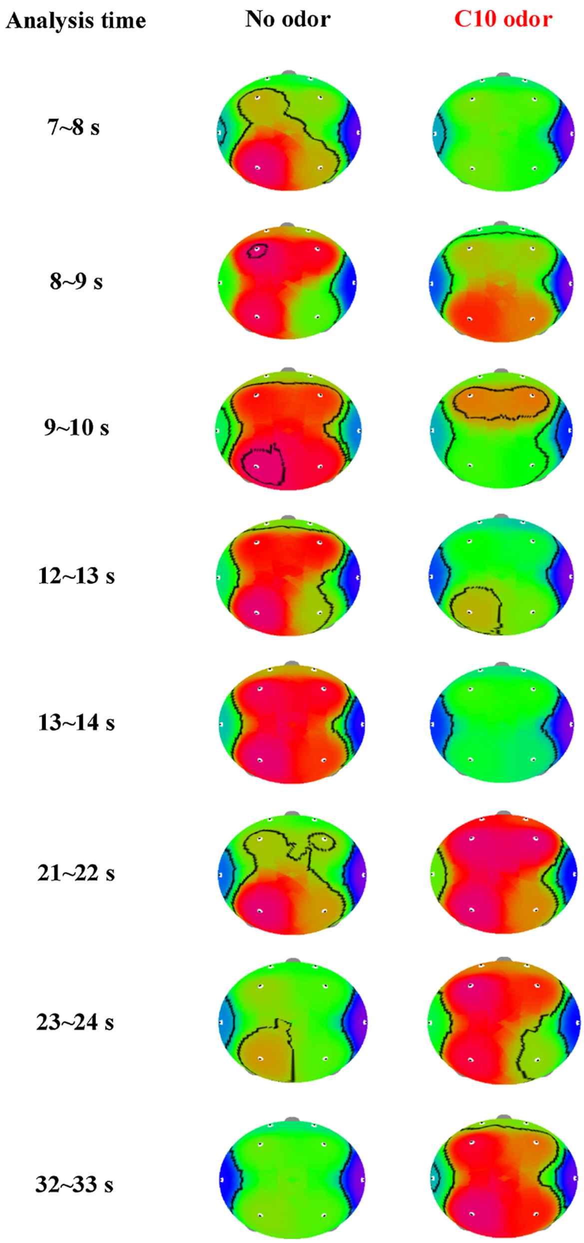

Fig. 2. The t-Mapping of absolute alpha (AA) power spectrum changes during no odor and C10 odor exposures according to time series analysis.

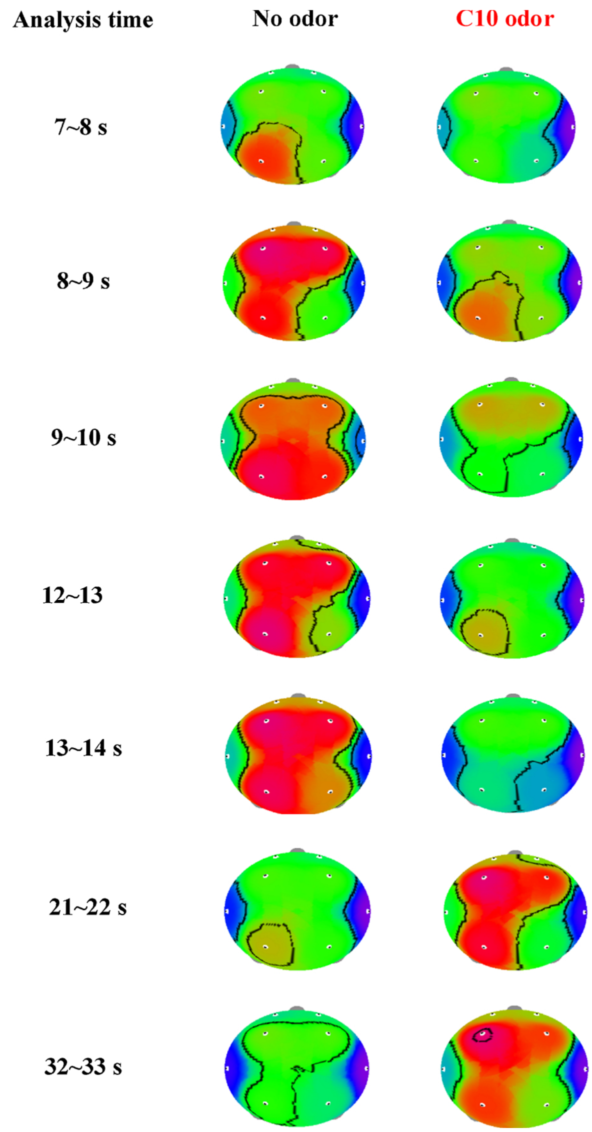

Fig. 3. The t-Mapping of absolute slow alpha (ASA) power spectrum changes during no odor and C10 odor exposures according to time series analysis.

prefrontal regions (Fp1 and Fp2) than temporal and parietal regions. In these, C10 odor highly produced significant changes at F3 region than other regions. In addition, significant changes of alpha and beta waves were mainly observed compared to that of theta and gamma waves. The time series data show that the exposure of C10 odor produced unstable EEG power spectrum changes.

4. Discussion

In the time series analysis, absolute waves such as theta, alpha, slow alpha, fast alpha, mid beta, low beta and gamma activities significantly decreased in different regions at the different time point during the first 13 s of C10 odor exposure with the exception of the beta wave. From 18 ~ 19 s, AA, ASA, AFA and AHB waves significantly increased, but there was no specific trend in AB, ALB and AMB waves due to the exposure of C10 odor (Table 2).

In this study, AA, ASA, and ALB waves mostly changed than other brain waves during the time series analysis (Fig. 1). There was no significant change in AG during the last 16 s of C10 odor exposure. In the brain, the electrical activity of neurons produces time-varying potential differences on the scalp. A study indicated that a significant decrease of alpha 1 was observed for lavender, eugenol, and chamomile (except sandalwood) during the first 10 s period of odor exposure. The significant decrease was persistent during the period of lavender odor exposure. Whereas the decrease was not persistent for the exposure of other odors [16]. Recently, some authors used the time-frequency analysis method to study EEG signals in both the time and frequency domains simultaneously. In the olfactory stimulation study, time-frequency analysis of the EEG activity can be effectively used for the evaluation of olfactory function in patients [17]. Schriever et al. [18] found that central processing of olfactory stimuli (phenylethyl alcohol and eucalyptol) analyzed by time-frequency analysis constantly differentiates patients with olfactory dysfunction from healthy individuals at a high degree of precision. According to functional magnetic resonance imaging study, the time course of odor molecules-stimulated activation in the human primary olfactory cortex is 30 ~ 40 s [19].

Among different brain waves, alpha and beta brain waves play an important role in the enhancement of various cognitive functions in human. Previous studies clearly suggest that the alpha wave activity is attenuated under emotional tension and stress states [9,20,21]. Overall,

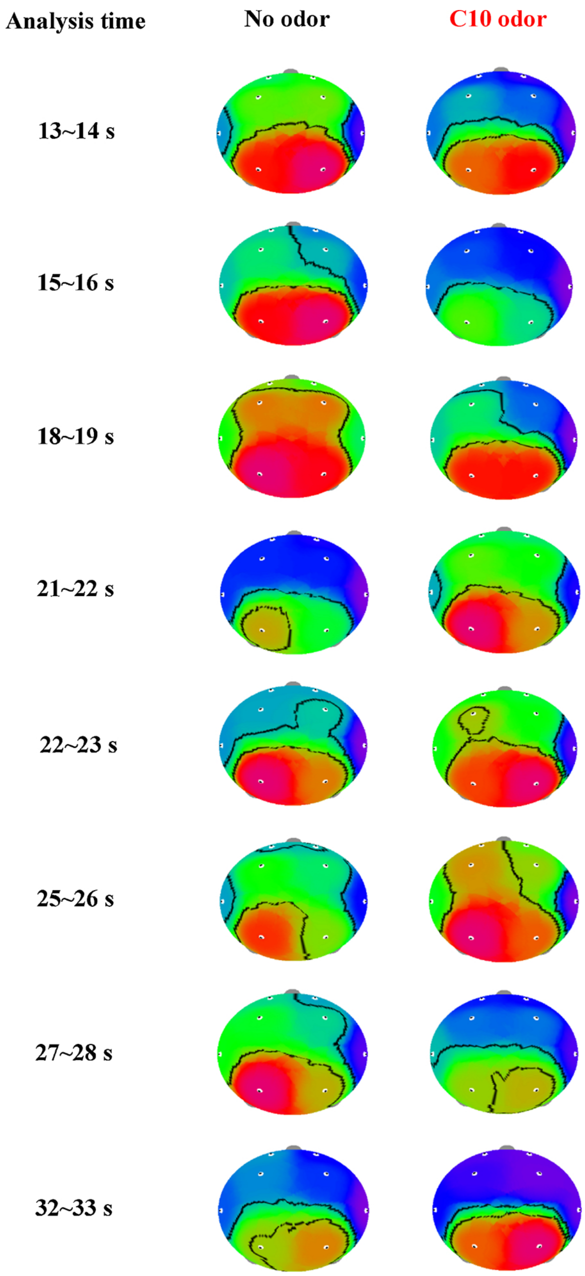

Fig. 4. The t-Mapping of absolute low beta (ALB) power spectrum changes during no odor and C10 odor exposures according to time series analysis.

the decrease of alpha wave activity due to the fragrance inhalation of C10 may increase the tension and stress states of the brain. However, the significant change of alpha waves (AA, ASA, and AFA) was totally opposite at a certain time point during the last 17 s. In the case of beta waves, AB, ALB, AMB, and AHB significantly decreased during the first 13 s and increased at some time point. But these beta waves did not show the same trend of increase during the last 17 s. In general, the decrease in beta wave activity is mainly associated with drowsiness state and the increase in beta wave activity is mainly associated with high awareness state of the brain [22].

Furthermore, a significant decrease of absolute theta wave activity (at 7 ~ 8, 9 ~ 10, 29 ~ 30 and 31 ~ 32) was observed during the inhalation of C10. The reduction in theta wave activity is highly linked with the formation of memory [23–25]. Therefore, the reduction of AT wave activity suggests that the odor exposure of C10 may enhance the memory formation. Based on the time series data, the first 13 s of C10 odor exposure may increase the stress state of the brain and the last 17 s of C10 odor exposure may enhance the relaxation state of the brain. The results of the present investigation clearly suggest that aldehyde C10 significantly produced changes in different EEG power spectrum values at different brain regions based on the time series analysis.

In addition, significant changes were mainly observed for alpha and beta waves compared to that of theta and gamma waves. Furthermore, the findings of the present study indicate that the fragrance inhalation of C10 highly affected the frontal regions followed by prefrontal regions. In particular, C10 odor highly produces significant changes at the left frontal region when compared to other regions. In the brain, the prefrontal and frontal regions are the most important functional areas. The frontal region has been mainly linked with a variety of cognitive functions such as planning, problem-solving, speech, intellect, behavior, attention, and movements [2,26]. The previous study demonstrated that odors with pleasant smell mainly activate the medial orbitofrontal cortex region of the brain [27]. In the olfactory process, olfactory receptor cells play a key role in the detection of various fragrances. Hence, the fragrant molecules first reach olfactory receptor cells and then activate primary olfactory cortex through the olfactory bulb [28]. Stimulation by any odor molecules mainly activates primary olfactory regions, including piriform cortex, amygdala and neighboring cortex [29]. The communication between inhaling and odor molecules may play a major role in the activation of the primary olfactory cortex [19].

The present study has some limitations. Firstly, the EEG was recorded for a short duration such as 45 s during no odor and 45 s during C10 odor conditions. Secondly, it is difficult to predict the exact psychophysiological function of C10 odor owing to its non-persistent EEG changes during time series analysis and more deviations in the EEG readings between the subjects. Thirdly, the C10 odor was sequentially administered to each subject. The lack of randomization is also a major limitation of the present study. In the light of these limitations, further studies are warranted in relation to different concentrations of an odor stimulus, randomization of odor presentation, and slightly longer duration of EEG recording time with time series analysis.

5. Conclusion

The increase in the value of absolute alpha waves is highly associated with the positive psychophysiological conditions of the human. The present study clearly demonstrates that the EEG activity of odors is highly influenced by data analysis time. Therefore, further studies are warranted in relation to EEG recording with different duration using time series analysis.

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Conflict of interest

The authors declare that they have no potential conflict of interest.

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